The Egyptian Hypertension Society is planning to prepare a new book containing its recommendations and guidelines for management of heart failure. The success of our two previous guideline books titled" Management of Hypertension" and "Management of Coronary Artery Disease" has stimulated the board of directors of the Egyptian Hypertension Society to approve, in its meeting on March 24th 1999, the establishment of a heart failure guidelines working group to prepare a new book titled "Heart Failure". The book will be addressing the practitioner, the internist and the cardiologist. It will contain the essential and modern information that will guide our Egyptian physicians in their everyday practice. Heart failure is possibly the most important cause of hospital admission to cardiology and general medical departments. Furthermore, developments in drug therapy in the past decade have changed favorably the course and outcome of this serious disorder in many patients. For a number of reasons heart failure has become an increasingly important problem in our clinical practice. First, there is a rising incidence of heart failure in the population, and it is expected that this trend will continue to increase in the coming years. The incidence of heart failure rises with aging. There is a worldwide increase in average life expectancy with an increase in the number of the elderly in many countries. Furthermore, the introduction in the recent years of effective therapies for hypertension, coronary artery disease and valvular heart disease has prolonged survival in many cardiac patients and delayed the development of heart failure.

Second, in the last two decades, a number of new therapeutic interventions were introduced for the management of heart failure. Many of these interventions not only improved patients' symptoms and quality of life but also prolonged survival of patients with heart failure and decreased rates of hospitalization and cardiovascular events. Angiotensin converting enzyme inhibitors (ACEI), beta adrenergic blockers, Angiotensin receptor blockers and spironolactone have now an established role in the routine management of heart failure patients. In addition, to these established drugs, new pharmacologic agents are under extensive investigation, which include neutral endopeptidase inhibitors (NEPI) (Candoxatrilat), combined NEPI and ACEI (Omipatrilat), endothelin receptors antagonists (Bosentan), natriuretic peptide (Nesiritide), tumor necrosis factor receptor antagonists (Etanercept), new classes of inotropic agents with a novel mode of action independent of the cyclic adenosine monophosphate (cAMP) pathway such as calcium sensitizers. These agents proved effective in improving the hemodynamics and effort tolerance in short term studies. The plethora of pharmacological agents combined with non-pharmacological interventions such as cardiac pacing, LV assist devices, surgery, gene and immunotherapy make the management of heart failure patients something more than simple digitalis, diuretics and bed rest.

Finally, the recognition that asymptomatic left ventricular dysfunction is a common condition, being the precursor of clinical heart failure in about one third of patients and the observation that ACEI therapy can delay the progression into overt heart failure, have two implications. First, that echocardiography should assume a central role as a screening tool for asymptomatic impaired LV function. Second, there are new insights into the possibility of prevention of heart failure through early intervention, particularly with the use of drugs (ACEI and beta blockers) in asymptomatic patients.

M. Mohsen Ibrahim M.D.
Prof & Chairman, Department of Cardiovascular Medicine-Cairo University
President of The Egyptian Hypertension Society

THE PRESIDENT'S MESSAGE
INCREASING IMPORTANCE OF HEART FAILURE

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changed favorably the course and outcome of this serious disorder in many patients. For a number of reasons heart failure has become an increasingly important problem in our clinical practice. First, there is a rising incidence of heart failure in the population, and it is expected that this trend will continue to increase in the coming years. The incidence of heart failure rises with aging. There is a worldwide increase in average life expectancy with an increase in the number of the elderly in many countries. Furthermore, the introduction in the recent years of effective therapies for hypertension, coronary artery disease and valvular heart disease has prolonged survival in many cardiac patients and delayed the development of heart failure.

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SCIENTIFIC NEWS

Unraveling some of the etiopathogenic aspects in hypertension cleared that a difference in endothelium activity was detected between black and white population and was suggested to play a major role in the clinical differences observed among them and the therapeutic outcome specially in relevance to the different antihypertensives available..

Novel therapies to treat cardiovascular diseases as hypertension are aimed at striking the critical steps in vascular disease progression. This includes reversing endothelial cell dysfunction, correcting dysregulated cell growth and Apoptosis ,modulating vascular phenotype, modifying mechano-transduction and reversing vascular remodeling.

CONTENTS

- Practical considerations:
- Hypertensive women
- Environmental hazards:
- Cigarette smoking
- Bed-side tips
- Cardiology pearls
- National & international
- recognition
- EHS news
- Calendar

EDITORIAL

INSULIN RESISTANCE AND ESSENTIAL HYPERTENSION

Mona Aboul-Seoud, MD
Endocrine Unit, Internal Medicine, Department,
Faculty of Medicine, University of Alexandria.

The past decades have witnessed a major surge of interest in the cardiovascular actions of insulin. On one hand, this interest has stemmed from epidemiological studies demonstrating an association between obesity, insulin resistance, and hypertension, which has been named the "insulin hypothesis of hypertension.' On the other hand, this interest has been stimulated by experimental evidence suggesting that the vascular actions of insulin contributes to its essential role in promoting glucose uptake in skeletal muscles.

In such a domain, two tenets have emerged about how insulin may exert its cardiovascular actions. Thus, it is firmly established that acute insulin administration stimulates sympathetic nerve activity in both animals and humans. Also, increasing evidence clear that insulin stimulates muscle blood flow, an effect that appears to be mediated, at least, in part, by an endotheliumdependent mechanism.

In this respect, insulin is thought to stimulate endothelial NO production or may act directly to enhance hyperpolarization of vascular smooth muscle cell membranes via stimulation of their Na+-H+ exchanger and Na+-K+ ATPase, leading to the consequent closure of their voltage-gated Ca2+ channels. While glucose uptake, may determine peripheral blood flow via stimulation of ATP-dependent ion pumps with consequent vasorelaxation.

Beyond this, a "third factor" may collectively contribute to both insulin resistance and endothelial dysfunction in cardiovascular disease. Candidates to this include; skeletal muscle fibre type and capillary density, distribution of adiposity and endogenous corticosteroid production.

The aformentioned can highlight how hyperinsulinemia that commonly associates hypertension in the metabolic syndrome-X, can perpetuate the functional and structural alteration in vessels characteristic to essential hypertension. This hyperinsulinemia is attributed to the presence of decreased insulin sensitivity, or insulin resistance, with consequent compensatory insulin secretion.

When the hypothesis of decreased insulin clearance present in hypertensive subjects and its contribution to hyperinsulinemia [independent of the degree of insulin resistance] was tested; it was found that essential hypertension is independently associated with decreased insulin metabolic clearance rate in addition to insulin resistance. Thus, a low insulin metabolic clearance rate may be a contributory factor to the hyperinsulinernia observed in essential hypertension.

The pathophysiological mechanisms linking hyperinsulinemia to hypertension are varied. Insulin might increase blood pressure through sympathetic nervous system stimulation and enhancement of renal sodium absorption. Evidence exists linking both of these mechanisms to hypertension. Also, insulin is independently associated with

myocardial infarction and microalbuminuria, two long-term complications of high blood pressure. While experimentally induced decreases in insulin resistance and hyperinsulinemia have been associated with decreased blood pressure. Moreover, normotensive offspring of hypertensive parents are also, as a group, insulin resistant and hyperinsulinemic.

The relationship between hyperinsulinemia, insulin resistance and hypertension is more marked in the obese, yet is present in lean hypertensive as well. However this relationship is not present in secondary forms of hypertension and may persist despite adequate antihypertensive therapy.

When the impact of therapeutic agents on insulin resistance were raised, their appeared that treatment of essential hypertension with beta-blockers and diuretics has been associated with increased risk of developing diabetes mellitus in three prospective cohort studies. While prospective, randomized studies with antihypertensive drugs have demonstrated differences between different classes of drugs regarding effects on insulin sensitivity. Accordingly, treatment with beta-blockers or diuretics is associated with impairment in insulin sensitivity, whereas most modern calcium channel blockers and Angiotensin converting enzyme (ACE) inhibitors are neutral. However, still to hold, there are exceptions within the different classes. For instance, Captopril seems to differ from other ACE inhibitors and results in improvement of insulin sensitivity yet the most pronounced improvements have been obtained with alpha1 blockers.

Despite of the raised therapeutic control, new concepts in therapy of insulin resistance are endlessly added, realizing how much this syndrome is rapidly expanding in industrialized countries, with its dramatic consequences on public health.

For instance, insulin sensitivity can be improved by non-pharmacological means as, the essential reduction of excessive body weight, the promotion of regular physical activity and the modification of dietary habits, as well as, the possibility of cessation of smoking and correction of subclinical magnesium deficiency. While the currently available pharmacological means should mainly include the biguanide compound metformin and possibly anti-obesity agents, such as dexfenfluramine, fluoxetine and benfluorex. New compounds aiming at improving the action of insulin, that are called 'insulin sensitizers" as the thiazolidinedione derivatives like troglitazone and pioglitazone are better added. By this therapeutic modality, hopefully the cardiovascular prognosis of numerous individuals having some or all components of the insulin resistance syndrome is improved.

In conclusion, evidence suggests that hyperinsulinemia and insulin resistance exert a pro-hypertensive effect that contributes to the pathogenesis of hypertension and hypertensive complications in some patients with essential hypertension. The associated complex interaction between endothelial dysfunction, abnormal skeletal muscle blood flow and reduced insulin-mediated glucose uptake observed, links between insulin resistance, blood pressure, impaired glucose tolerance and the risk of cardiovascular disease. An understanding of the primary mechanisms resulting in these phenotypes may reveal new targets to therapeutically strike when tempting to achieve appropriate control. While in those at high risk for diabetes mellitus, it may be justified to select drugs that improve insulin sensitivity when treating hypertension in insulin resistant individuals

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ABSTRACTS OF WORLD LITERATURE
ECHOCARDIOGRAPHIC DEFINITION OF LEFT VENTRICULAR HYPERTROPHY IN THE HYPERTENSIVE: WHICH METHOD OF INDEXATION OF LEFT VENTRICULAR MASS?

P Gosse, V Jullien, P Jarnier, P Lemetayer & J Clementy
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Objectives: It has been suggested that hypertensives at high risk of cardiovascular complications can be identified on the basis of their left ventricular mass as determined echographically. However, there is as yet a lack of consensus on the mode of indexation (body surface area, height, height2.7) of left ventricular mass (LVM), and on the cut-off values for definition of left ventricular hypertrophy (LVH). The main objective of this study is to test the influence of the different modes of indexation for LVM on the prevalence of LVH in a population of never treated hypertensive patients on the basis of cut-off for LVM based upon its relationship with ambulatory blood pressure (BP)measurement.

Methods: A population of 363 untreated hypertensives was investigated using a standardised procedure. The men and women were analyzed separately. We studied the relationship between mean daytime ambulatory systolic BP and LVM and calculated the LVM cut-off for a BP of 135 mm Hg using three different methods of indexation. On the basis of these criteria, the population was divided into those with and those without LVH.

Results: Th prevalence of LVH was found to be higher when LVM was indexed to height 2.7 (50.40o) or height (50.1%). Prevalence was lowest when LVM was indexed to body surface area (48.20o), which tended to minimize the hypertrophy in obese individuals. Only indexation by height2.7 fully compensates for relationships between height and ventricular mass in this population.

Conclusions: Indexing LVM to height 2.7 thus appeared to give a more sensitive estimate of LVH by eliminating the influence of growth. Cut-off of 47 g/m 2.7 in women and 5 g/m2.7 in men corresponded to a cardiovascular risk indicated by a daytime systolic BP 135 mm Hg.

I Hypertension 1999;13 (8): 559- 563.

TRIALS OF ANTIHYPERTENSIVE THERAPIES IN CHILDREN.

Wells TG
Divisions of Pediatric, Nephrology and Pediatric Clinical Pharmacology and Toxicology, University of Arkansas for Medical Sciences and the Arkansas Children's Hospital, Little Rock, Arkansas, USA.

Clinical trials assessing the safety, effectiveness and pharmacokinetics of new antihypertensive medications have been numerous as new classes of medications have been developed and brought to market over the past two decades. However, very few clinical trials have been initiated and completed in children with hypertension. Excluding diuretics, only one antihypertensive medication marketed within the past 20 years has any pediatric pharmacokinetic or dosing information published in the drug label and none have a pediatric indication. There are many reasons that these studies have not been done. Summation of the data collected in large epidemiologic studies that establish normal blood pressure and define hypertension using casual measurements have been a relatively recent event in pediatrics. Although ambulatory blood pressure measurement has been studied for the past decade there is still uncertainty with respect to the standardization of devices, measurement technique and normal values in a multi-racial pediatric population. As a result, no large scale, industry-sponsored clinical trials involving antihypertensive therapy have employed this measurement technique in children. In recognition of this problem, US Congress passed the Food and Drug Administration Modernization Act in 1997. Among the many provisions of this law, the US Food and Drug Administration (FDA) is required to publish a list of approved drugs for which additional

information may prove beneficial for children. This law and subsequent action by the FDA also provides a mechanism by which manufacturers may gain six months of additional market exclusivity if adequate and well-controlled pediatric trials are completed and submitted to the FDA in response to a formal written request for these studies. Because such studies have not been previously undertaken and the new rules provide a significant financial incentive, written requests have been issued for pediatric studies involving more than a dozen antihypertensive agents. The FDA published a sample written request for oral antihypertensives in children and several potential study designs were presented.

Blood Press Monit l999;4(3):189-192

ABSTRACTS OF LOCAL LITERATURE

PRO INFLAMMATORY CYTOKINES IN ISCHEMIC HEART DISEASE: EFFECT OF CONVERTING ENZEME INHIBITORS.

Saraa Abd EI-Sha.fee, Ahmed Abd El-Aziz,, Abd EI-Hameed Ibrahim, Abeer Mohamed, Sobhy Said, Sherif Mokhtar.

Critical Care Medicine, Cairo University & Clinical Pharmacology, Helwan University.

Pro-inflammatory CYTOKINES are mediators released from leucocytes and vascular endothelium in response to various inflammatory and noxious stimuli, but have recently been reported also in myocardial ischemia and different grades of heart failure (HF). The exact pathogenic role and mechanism of production in HF and Ischaemic heart disease have not been clearly explained. Moreover the role of converting enzyme inhibition has not been elucidated in this work, we assessed the level of Cytokines and studied the effect of Angiotensin converting enzyme (ACE) inhibition on such mediators using Captopril (75 mglday) and Benazepril 5-10 mg /day in 2 groups of pts with ischeamic heart disease (IHD). Group 1: comprized 16 pts with chronic myocardial ischemia (12 males, mean age 49± 15) all complicated by heart failure (class 11 NYHA). Group 11: comprized 20 pts (17 males, mean age 52 - 10) with acute myocardial infarction complicated by left ventricular dysfunction (Killp's classification 11). Following admission all patients had clinical and laboratory evaluation including serum estimation of Cytokines I L6 and 1 L8 measured by chemiluminescence immunometric assay technique. Converting enzyme inhibition was started in the absence of contraindication using Captopril and Benazepril for one week with repeated measurement of serum Cytokines. Compared to the group of pts with chronic IHD, those with acute myocardial infarction had significantly higher levels of interleukin 6 (180 vs 10 pg/rn) and interleukin 8 (400 vs 20 pg/rn), respectively prior to treatment with ACE inhibitors. Following ACE therapy there was significant reduction in interleukin 1L6 (from 180 to 20 pg/ml) and 1L8 (from 400 to 90 pg/mI) in groupll. We concluded that pro-inflammatory Cytokines are elevated in acute MI apparently as a response to the acute Ischaemic insult rather than being related to heart failure. More important, converting enzyme inhibitors were shown for the first time to significantly reduce the pro-inflammatory Cytokines in acute myocardial infarction a further addition to their cardiac protective action. Presented at the 3rd Scientific meeting of the Egyptian Hypertension Society, Port Said, Egypt. December 1998.

BETA ADRENOCEPTOR MEDIATED VASORELAXATION OF RABBIT AORTA IS ENDOTHELIUM-DEPENDENT: ROLE OF PROSTAGLANDINS AND SODIUM PUMP

Hassan Heialy Abo Rahma
Department of Pharmacology, Assiut Faculty of Medicine, Assiut, Egypt

B-adrenoceptor mediated vasorelaxation is thought to be through activation of adenylate cyclase within the smooth muscles. Such a mechanism is not dependent on the presence of the intact endothelium. There are now several findings suggesting that the B-adrenoceptor induced vasodilatation is endothelium dependent. However, others have shown that isoprenaline-induced relaxations in rat aorta are not endothelium dependent. Moreover, there is accumulated evidence showing that hyperpolarization through opening of K-ATP channels partly mediates vasodilatation induced by B-adrenoceptors. Recently, it was reported also that nitric oxide mediated Na+ pump as well as cyclooxygenase activation are also involved. The present study was designed to evaluate the role of endothelium in 3-adrenoceptor mediated vasorelaxation in isolated rabbit aortic rings by using the selective 32

agonist, terbutaline and to investigate the possible involvement of other mediators e.g. prostaglandins and other cellular components e.g. Na+-K+ ATPase (sodium pump) in the

B-adrenoceptor mediated vasorelaxation. The results of the present work indicate that the removal of the endothelium reduces significantly the relaxant effect of terbutalin Propranolol treatment reduced significantly ($p< 0.05$) the terbutaline induced vasorelaxation & shifted its cumulative dose-response curve to the right. Terbutaline concentration response curves were obtained after treatment of the aortic rings with the prostaglandin synthesis inhibitors, dexamethasone and aspirin. The results showed that the terbutaline induced vasorelaxation was significantly augmented ($p<0.0l$) in presence of dexamethasone. This means that not only the prostaglandins are involved but also other substances e.g. adenosine, leukotrienes, etc. may be involved and the net result depends on the proportion of the vasodilator and the vasoconstrictor agents that may be affected by the cyclooxygenase and the phospholipase A2 inhibitors. Pretreatment of the aortic rings with the sodium pump inhibitor, digoxin, produced a significant reduction ($p<O.01$) of the terbutaline induced vasorelaxation: pretreatment with dexamethasone, aspirin and digoxin didn't affect significantly the endothelium independent sodium nitroprusside induced vasodilatation. In conclusion, B-adrenoceptor vasorelaxation is mediated partly by NO release from the endothelium. The results of the present work suggests also the involvement of other mediators e.g. prostaglandins and the sarcolemmal sodium pump in the B-adrenoceptor mediated vasorelaxation of the rabbit aorta

Presented at the Joint International Conference of Egyptian Society of Pharmacology & Experimental Therapeutics, the Union of African Societies of Pharmacology & the Arab Union of Pharmacology. Cairo, Egypt, December 1999
CHALLENGE YOUR SELF !!!

A 73-years old man with known hypertension for many years was admitted for evaluation of progressive exertional dyspnea that developed over several days and for the sudden onset of severe dyspnea. He denied recent chest pain but did note that 1 year earlier he had experienced several episodes of nonspecific chest pain. He underwent coronary arteriography at that time, which demonstrated normal coronary arteries. He had no history of diabetes and did not smoke.

Physical Examination: Vital signs; pulse; 1 00;BP 220/120. General: severe dyspnea. Neck: no venous .distension. Chest: crackles throughout all lung fields. Cardiac: no S3 or murmur. Laboratory investigation; Cardiac enzymes: normal. Chest radiography: pulmonary edema with normal heart size. EKG: normal. Echocardiogram and Doppler study: normal chamber sizes with some left ventricular hypertrophy and an ejection fraction of 600 o; mitral flow revealed a reduction of the F to A ratio.

Question: What is the cause of this patient's acute pulmonary oedema?
Pick up the solution at CARDIOLOGY PEARLS
on p. [7] of this issue. PRACTICAL CONSIDERATIONS:

HYPERTENSIVE WOMEN

When your patient happens to be a woman please;

àConcentrate on the ways to make this patient day by day live healthier and feel better and emphasize that it's a task every woman can do.

à Explain the dimension of the problem first and clear that three of every four women with high blood pressure know they have it, yet fewer than one in three are controlling it.

àEncourage overweight patients to a gradual loss of weight either by a lower calorie intake or increased physical activity and preferably both... Advice from a registered dietitian, or a qualified nutritionist to plan for a sensible, balanced eating pattern to lose weight slowly should be encouraged.

àExplain the importance of physical activity for the heart and blood vessels and for maintaining optimal weight to

look and feel better. Luckily, this can be achieved without having to run marathons. Just a 30 minutes of moderate activity would be a brisk walking, gardening, cycling, swimming... .Breaking this into periods of at least 10 minutes, helps one to get started. If the issue that your patient is pressed for time, just make it clear that physical activity doesn't need that much time for a reward and a sense of accomplishment.

àExplain that women, should have no more than 2,400 milligrams of sodium a day (about 1 teaspoon) and clear, that this amount includes all of the salt and sodium consumed in processed foods, added during cooking, and used at the table. The importance of this hold of salt, is equal, whether there is high blood pressure, high-normal blood pressure, or to prevent high blood pressure.

àWhen you prescribe an antihypertensive, be sure that your patient understands the instructions and secure that if something is not clearly understood, she should call back and ask. Explain that, as with all drugs, side effects like; sleepiness, being tired, rash or cough etc, could happen. So it is important that she should pay attention to how she feels and notify any change that issues.

--Guidelines to which antihypertensives allowed during pregnancy, lactation, with contraception or better avoided in special health problems will be highlighted in the forthcoming editions.

High Blood Pressure Information for the General Public
: NHLBJ Home Page A collaboration of the Alliance for Aging Research,
National Heart, Lung, and Blood Institute, National
Institutes of Health & Sponsored by Hoechst Marion
Roussel, Inc.

ENVIRONMENTAL HAZARDS:
CIGARETTE SMOKING: A GLOBAL LOOK

Many epidemiological and experimental studies has recognized and established the links between cigarette smoking and its cardiovascular hazardous impacts on morbidity & mortality.

Speaking of its relevance to hypertension per se, it was reported that smoking a cigarette raises the blood pressure by 5-10 mm Hg for about 30 minutes and lowers coronary reserve for 20 minutes. This aggravates and lasts longer if this is combined with drinking a cup of coffee.

Despite of this, numerous epidemiological studies have found that people with hypertension are not more likely to be smokers than those with normal blood pressure, and conversely that smokers are not more likely to be hypertensive than non-smokers. One possible explanation for this, might be, that smokers tend to weigh less than non-smokers, and that the effects of obesity and smoking on blood pressure cancel each other out. But even when smokers and nonsmokers of the same body weight are compared, their blood pressures are the same. This is probably because the rise of daytime pressure, after one smokes a pack a day, will raise your average pressure by about 5 mm Hg, a value that might not be detected during an office visit to the physician.

However, the important thing about smoking, is not what it does solely to your blood pressure, [the mechanisms through which remains to be clarified] but is centered more to how it greatly increases the vascular and cardiac risks, the extent of end organ damage and the likelihood of co-morbid conditions. These issues then, will even weigh more.

Moreover, breathing sidestream smoke [passive smoking] is also partially associated with the same detrimental effects as active smoking. To imagine how figures could be striking, the U.S. Environmental Protection Agency estimated that in USA 30,000 to 60,000 cardiac deaths are associated with passive smoke exposure. The question that will pose itself at this juncture is: Will these risks go down, if one quit smoking? The answer will be a big ' YES". This statement is based on the findings of several studies clearing that if you smoke, your risk of heart disease is about three times higher than if you don't. If you quit, it will go down to twice the normal risk in two years, and

after that, it will be the same as if you had never smoked.

Realizing all this, calls on the need to really under stand why and how cigarette smoking does all this.
So let us know first, what are some of the noxious chemicals in tobacco smoke???
It was reported that tar, phenol, benzopyrene, nirtosamine and polycyclic aromatic hydrocarbons are all carcinogenic and some are pro-oxidants thus perpetuating lipid peroxidation, endothelial disruption and impairing NO vasodilatory potentials, which are all relevant to hypertension and comorbid atherosclerotic changes. While formaldehyde and oxides of nitrogen are severe bronchial irritants and toxic to epithelial, endothelial,.. .etc structures. In addition, carbon monoxide impairs oxygen transport and utilization and shares in a pattern of Ischaemic myocardial insults that may also be linked to microvascular ischaemias in hypertensive heart disease. Lastly nicotine the known ganglion stimulant and depressant, beyond being carcinogenic, it sets the sympathetic drive of blood pressure regulation, at a higher regulatory setpoint.

The impact of some of such hazardous products and there relevance to vascular and cardiac functional and structural integrity will be more detailed in the forthcoming editions.

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N Engl J Med 1996; 334: 1189.
J Hum Hypertens 1996 Feb;10 Suppl 2:S13-6
Basic Pathology. W.B . Saunders Company
Publicationsl997; 8: 221.

PATIENT'S TIPS:

Help your patients stop smoking by initiating therapy with the antihypertensive mecamylamine HCI [Inversine] 10 days before their quit day starting by 2.5 mg twice daily for five days & going up to 5 mg twice a day. Mecamylamine reduces nicotine craving because it blocks ganglionic receptors. So tell the patient to stop smoking completely & start wearing the nicotine patch. By this you can strike a double benefit ; cessation of smoking & improving the therapeutic outcome of any antihypertensive intended to be used.

Modem Medicine 1998; 15 : 32.

CARDIOLOGY PEARLS

1. Diagnosis: Pulmonary oedema secondary to hypertension with diastolic dysfunction.
2. One-third of patients with signs of congestive heart failure have diastolic dysfunction and normal systolic function.
3. Diastolic dysfunction should be suspected in a patient with signs and symptoms of congestive heart failure who has normal-sized heart and normal ejection fraction.
4. The differentiation of systolic versus diastolic dysfunction is important, as therapy is different for these two causes of heart failure. Inotropic agents and arterial vasodilators used for unloading therapy may be harmful in diastolic dysfunction

NATIONAL & INTERNATIONAL RECOGNITION:

- Prof Dr. Mohsen Ibrahirn, Head of Dept. Cardiology, Faculty of Medicine, Cairo University and the President of the Society, has been chosen a member of the Editorial Board for the Journal of Hypertension, for a term of three years. This journal happens to be the official Journal of the International Society of Hypertension (SH) and the European Society of Hypertension (ESH).
- Prof. Dr. Ebtihag A Hamdi, Prof in Cardiology Unit, Faculty of Medicine, Alexandria University and a senior member of the editing board of this News Letter has been elected as a member of the High Blood Pressure Council, the Scientific Council of the American Heart Association, starting from 1st June 1999

with a membership ID 000106559901

EHS News & Calendar

EHS NEWS:

- The EHS has held its annual social event of getting together in Ramadan at Cairo Sheraton Hotel on Friday the 24th of this month. The members enjoyed the splendid iftar and the whole heartily gathering in this holy month . Prof Dr. Mokhtar Gomma clarified the final arrangements and theme of organization he has adopted in the forthcoming annual congress of the society next January. Also, Prof Dr. Soliman Gareeb highlighted the preliminary data pooled from the scientific committee of the Egyptian Hypertension Project that is conducted to test the efficacy and tolerability of anti-hypertensives among Egyptian hypertensive population. A short statement was addressed by Prof Dr. Omnia Nayel on the current status of the news letter of the society an the topics that has been added to the issues. Lastly Prof Dr. Mohsen Ibrahim delivered a small lecture to delineate the criteria that should be fulfilled when probing the utility of an antihypertensive agent He also handed out a questionnaire form termed " Physician Hypertension Survey" to investigate Egyptian physician's knowledge and attitudes regarding hypertension.

CALENDAR:

LOCAL MEETINGS

$th Meeting of the Egyptian Society of Atherosclerosis	Mena-House Oberoi,Cairo Egypt. January 21-23, 2000.	Prof Dr. Osama Abdel Aziz Tel: (202) 3926650, Telefax: (202) 3602800 / 3958000
The fourth Scientific Meeting of the EHS	Marriot Hotel, Cairo Egypt January 28-29th 2000	Prof Dr. Mokhtar Gomma Tel: (202) 3026871 -Fax (202) 3026871 E-mail : mogomaa@idscl.gov.eg
7th Annual Meeting of Egyptian Society of Cardiothoracic Surgery	Sheraton El Gizzera Hotel Cairo,Egypt, February,9-11,2000	Prof Dr.Shabaan Abul-Ela Tel : (2050) 374111.Fax (2050) 360138 E-mail : SHAABANABUELA@netscape.net
The 27th Annual Congress of the Egyptian Society of Cardiology	Marrior Hotel ,Cairo Egypt February 21st -25th ,2000.	Prof Dr.Adel Imam Tel (202) 3489383-Fax (202) 3489383 E-mail : emam@internetegypt.com

INTERNATIONAL MEETINGS

4th World Congress of Echo-cardiography & vascular Ultrasound.	Mena-House Oberoi,Cairo. Egypt. January 19-2 1, 2000.	Prof Dr. Osama Abdel Aziz Tel : (202) 3926650, Telefax: (202) 3602800 / 3958000
3rd Conference of the Pan -Arab Hypertension Society	Abu Dhabi, 5-9 February, 2000 Intercontinental Hotel	Conference Secretarial Office: Tel:+ 97 1(2) 347478-Fax 349225 E-Mail: yassinl@emirates.net.ae
9th International Congress on Cardiovascular Pharmacotherapy	Salvador, Bahia, Brazil. March 2000	Congress Secretariat; JZ Promocoes, E Assessproa De. Tel.: +55215391 299, Fax: + 55215379134

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Heart failure is characterized by the heartâ€™s inability to pump an adequate supply of blood to the body. Without sufficient blood flow, all major body functions are disrupted. Heart failure is a condition or a collection of symptoms that weaken your heart. In some people with heart failure, the heart has difficulty pumping enough blood to support other organs in the body. Other people may have a hardening and stiffening of the heart muscle itself, which blocks or reduces blood flow to the heart.Â Early treatment increases your chances of long-term recovery with fewer complications. Call your doctor right away if youâ€™re having any symptoms of heart failure. What are the symptoms of heart failure? The symptoms of heart failure may include Figure 8. Diagnosing heart failure. Figure 9. Unmet needs in the prevention, diagnosis, treatment and long-term management of heartfailure.Â 3. Improving public awareness of heart failure Public awareness of heart failure symptoms is dangerously low A healthy lifestyle reduces the risk of heart failure. 4. The need to apply best practice Guidelines worldwide agree on the key stages in heart failure care Best practice in heart failure care involves compliance with guidelines Encouraging compliance: measuring and improving quality of care. 5. Future directions in care: urgent unmet needs Diagnosis: improved tools for medical decision-making in heart failure Treatment: new options are needed for many patients with heart failure Long-t Chronic heart failure (CHF) is a multi-organ disease with increasing evidence for the involvement of the gastrointestinal (GI) system in this syndrome. In recent research, the gut has received very little attention from cardiologists as its role in the pathogenesis of cardiovascular disease is poorly understood. Intestinal ischaemia may play an important role in bacterial translocation by increasing bowel permeability. Decreased cardiac function can reduce bowel perfusion and so clearly impairs the function of the intestinal barrier. There is an increasing evidence to suggest that a 'leak Diabetes and heart failure are closely related: patients with diabetes have an increased risk of developing heart failure and those with heart failure are at higher risk of developing diabetes. Furthermore, antidiabetic medications increase the risk of mortality and hospitalisation for heart failure in patients with and without pre-existing heart failure. When the two diseases are considered individually, heart failure has a much poorer prognosis than diabetes mellitus; therefore heart failure has to be a priority for treatment in patients presenting with the two conditions, and the diabetic p President of Russia Vladimir Putin: Mr. President, Mr. Secretary-General, colleagues, ladies and gentlemen, This year, the international community celebrates two, without exaggeration, historic anniversaries: the 75th anniversary of the end of the Second World War and establishment of the United Nations. The importance of these two forever interlinked events cannot be overemphasized. In 1945, Nazism was defeated, the ideology of aggression and hatred was crushed, and the experience and spirit of alliance, as well as the awareness of the huge price that had been paid for peace and our common Vi...Â Message preview. Official Website of the President of Russia: 75th session of the UN General Assembly.